Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

Environmental Pollution 265 (2020) 115126

Contents lists available at ScienceDirect

Environmental Pollution

journal homepage: www.elsevier.com/locate/envpol

COVID-19 prevalence and fatality rates in association with air pollution emission concentrations and emission sources☆

Michael Hendryx [a,*], Juhua Luo [b]

[a] Department of Environmental and Occupational Health, School of Public Health, Indiana University, 1025, E. 7th St., Bloomington, USA
[b] Department of Epidemiology and Biostatistics, School of Public Health, Indiana University, Bloomington, IN, USA

ARTICLE INFO

Article history:
Received 25 April 2020
Received in revised form
25 June 2020
Accepted 26 June 2020
Available online 27 June 2020

Keywords:
COVID-19
Air pollution
Hazardous waste sites
Diesel particulate matter

ABSTRACT

The novel coronavirus disease (COVID-19) is primarily respiratory in nature, and as such, there is interest in examining whether air pollution might contribute to disease susceptibility or outcome. We merged data on COVID-19 cumulative prevalence and fatality rates as of May 31, 2020 with 2014–2019 pollution data from the US Environmental Protection Agency Environmental Justice Screen (EJSCREEN), with control for state testing rates, population density, and population covariate data from the County Health Rankings. Pollution data included three types of air emission concentrations (particulate matter<2.5 μm (PM2.5), ozone and diesel particulate matter (DPM)), and four pollution source variables (proximity to traffic, National Priority List sites, Risk Management Plan (RMP) sites, and hazardous waste treatment, storage and disposal facilities (TSDFs)). Results of mixed model linear multiple regression analyses indicated that, controlling for covariates, COVID-19 prevalence and fatality rates were significantly associated with greater DPM. Proximity to TSDFs was associated to greater fatality rates, and proximity to RMPs was associated with greater prevalence rates. Results are consistent with previous research indicating that air pollution increases susceptibility to respiratory viral pathogens. Results should be interpreted cautiously given the ecological design, the time lag between exposure and outcome, and the uncertainties in measuring COVID-19 prevalence. Areas with worse prior air quality, especially higher concentrations of diesel exhaust, may be at greater COVID-19 risk, although further studies are needed to confirm these relationships.

1. Introduction

The novel coronavirus disease (COVID-19) is an infectious disease characterized by respiratory illness that can range from mild to serious symptoms and death (WHO, 2020a). Common symptoms include fever, insistent cough, fatigue, sore throat, body pain, and shortness of breath. The disease outbreak began in December 2019, and as of June 4, 2020, there were over 6.5 million confirmed cases around the world and more than 387,000 confirmed deaths (WHO, 2020b). The United States has been particularly impacted by the disease, with more than 1.8 million confirmed cases and more than 107,000 deaths as of June 4, 2020 (CDC, 2020).

COVID-19 is primarily a respiratory disease, and as such, it has been of interest to understand whether and how air pollution might be a contributing factor to disease susceptibility or outcome (Contini and Costabile, 2020). Conticini et al. (2020) suggested that air pollution might have been a contributing factor to the high number of COVID-19 fatalities in Italy, and Ogen (2020) reported that high ambient levels of nitrogen dioxide (NO_2) were associated with COVID-19 mortality in Italy and Spain. Zhu et al. (2020) reported associations between particulate matter<2.5 μm and <10 μm (PM2.5 and PM10), NO_2 and ozone (O_3) and counts of confirmed COVID-19 cases in China. In the United States, Wu et al. (2020) presented results as of April 5 from a county analysis showing higher COVID-19 fatality rates in association with PM2.5.

We may consider possible mechanisms for associations between air pollution types or sources and COVID-19 susceptibility. Contaminated groundwater from hazardous waste sites can migrate through soil and into buildings via vapor intrusion, resulting in indoor air contamination (Johnston and MacDonald Gibson, 2015). Hazardous waste sites are also sources of air pollution (Fowler et al., 2010; Porta et al., 2009). There is evidence that air pollution, including PM2.5, NO_2 and diesel PM increases susceptibility to bacteria and viruses in the respiratory system (Castranova et al., 2001; Chauhan and Johnston, 2003; Jaspers et al.,

☆ This paper has been recommended for acceptance by Payam Dadvand.
* Corresponding author.
E-mail address: hendryx@indiana.edu (M. Hendryx).

https://doi.org/10.1016/j.envpol.2020.115126
0269-7491/

2005; Yang et al., 2001; Yang et al., 2020). Persons with pre-existing respiratory conditions are most susceptible. Diesel exhaust particles result in oxidative stress and inflammation in lung cells (Brandt et al., 2020; Takizawa et al., 2000). Long-term exposure to air pollution impairs lung function (Chauhan and Johnston, 2003; Gotschi et al., 2008) and may reduce resistance to viral infection (Yang et al., 2020). Long-term traffic-related pollution is one particularly important exposure source (Gotschi et al., 2008). Viruses normally are destroyed by phagocytosis, by which macrophages inhibit viral replication and remove virus-containing cells. Cytotoxic T-lymphocytes destroy infected cells. These normal functions can be impaired by exposure to air pollutants (Chauhan and Johnston, 2003).

Among prior studies on this topic, most have focused on fatality as the outcome and have examined several commonly studied air pollutants. Studies of disease prevalence are less common. Studies of possible pollution sources that may contribute to disease risk have not been reported. The purpose of the current exploratory, hypothesis-generating study was to examine associations between air pollutants that may represent long-term exposure, possible pollution sources, and prevalence and fatality from COVID-19 in the United States as of May 31, 2020. We included a measure of diesel particulate matter that previous studies on this topic have not included, and examined several possible pollution sources that have not been previously considered. We examined associations between pollution as measured by both emission concentrations and source exposure, and population prevalence and fatality from COVID-19.

2. Methods

2.1. Design and population

We conducted an analysis of COVID-19 cumulative prevalence and fatality per 100,000 population across 3143 US counties as of May 31, 2020. Pollution data were based on indicators from 2014 to 2019 as described below, such that associations represent possible long-term but not acute exposures. Pollution data were from the US Environmental Protection Agency (EPA) environmental justice screening tool EJSCREEN (EPA, 2018a). These data were merged with COVID-19 prevalence and fatality data obtained from the John Hopkins database (JHU, 2020) at the county level with control for county population density (persons per square mile), state-level testing rates, and population covariates from the County Health Rankings data (CHR, 2020).

2.2. Measures

2.2.1. Exposures

Exposure data were drawn from the US Environmental Protection Agency EJSCREEN (EPA, 2018a). Data for the most recent release year, 2019, were provided at the level of the Census block group, however, time of original data collection for the individual measures occurred earlier as shown below. We examined three measures of pollutant concentrations and four measures of pollution sources as defined in the EPA's technical manual (EPA, 2019a).

- Particulate matter: annual 2016 average PM2.5 concentration in micrograms per cubic meter (μg/m3).
- Ozone: Summer (May–September) 2016 average of daily maximum 8-h concentration in parts per billion.
- National Air Toxics Assessment (NATA) diesel particulate matter (DPM): 2014 average micrograms per cubic meter (μg DPM/m3) from all mobile emissions sources (EPA, 2018b).
- Traffic proximity: 2017 count of vehicles per day within 500 m of a block centroid, divided by distance in meters, presented as population-weighted average of blocks in each block group.
- Proximity to National Priorities List (NPL) sites: Count of 2019 sites proposed and listed on the NPL (a key subset of all Superfund sites), within 5 km of the average block group resident, divided by distance, calculated as the population-weighted average of blocks within each block group.
- Proximity to hazardous waste Treatment, Storage or Disposal Facilities (TSDFs): Count of 2019 TSDFs within 5 km, divided by distance, presented as population-weighted averages of blocks in each block group.
- Proximity to Risk Management Plan (RMP) sites: The 2019 count of RMPs (facilities required by the Clean Air Act to file risk management plans) within 5 km, divided by distance, presented as population-weighted averages of blocks within each block group.

To examine the effect of PM without diesel PM, we calculated an additional exposure measure as the difference between total PM2.5 and DPM.

2.2.2. Outcomes

The cumulative number of confirmed COVID-19 cases and number of confirmed deaths as of May 31, 2020 was obtained for every US county (N = 3143) (JHU, 2020). These counts were converted to counts per 100,000 county population using the population variable from the 2019 County Health Rankings data.

2.2.3. Covariates

Control variables from the County Health Rankings data (CHR, 2020) included percent of the population over age 65; percent race/ethnicity groups (African American, Asian, Native American/Pacific Islander, Hispanic, and non-Hispanic White (used as the referent in regression models)); percent of adults with at least some college education; income inequality (the ratio of household income at the 80th percentile to that at the 20th percentile); adult smoking rate; adult obesity rate; and percent of the population without health insurance. See the County Health Rankings website (CHR, 2020) for measurement details. Population density was measured from US Census data as population per county square mile, and number of COVID-19 tests per 100,000 state population was obtained from the COVID Tracking Project (2020).

2.3. Analysis

EJSCREEN data at the block group level were weighted by block group population and then summarized to the mean value at the county level. Each of the seven EJSCREEN indicators was standardized to a common scale with mean = 100 (SD = 10). Descriptive statistics for study variables were calculated and bivariate correlations examined. Mean imputation was used to replace missing values for income inequality (n = 2 counties), median household income (n = 1 county), and percent rural population (n = 7 counties.) Missing values for ozone and PM2.5 reduced the sample to N = 3108 for analyses with these variables.

Mixed model linear multiple regression analyses were conducted using SAS software version 9.4 Proc Mixed. Each model included covariates as described above. A state class variable was added as a random effect to account for possible within state correlations between counties. Separate models were first run for each of the seven EJSCREEN variables, followed by a model that included the three pollutant emissions simultaneously, and another that considered the four source measures simultaneously. Final models included all seven indicators simultaneously. Models were

repeated for COVID-19 prevalence and fatality rate per 100,000.

3. Results

A summary of study variables is provided in Table 1, categorized by COVID-19 prevalence (none, greater than zero and less than median, and higher than median). As of May 31, 2020, the mean county level COVID-19 prevalence was 313.39 per 100,000 population (range 0–12,640.76). The mean county level death rate per 100,000 was 12.72 (range 0–1324.31). Counties with prevalence rates above the median, compared to counties without cases, were younger, had higher percentages of African Americans and Hispanics, and higher smoking and obesity rates. Population density was greatest in areas with rates above the median, but was lowest in areas with positive cases below the median. Pollution emission concentrations (except ozone) were higher in the areas with higher disease rates.

A Pearson correlation matrix among the eight environmental indicators was provided in Supplemental Table 1. PM2.5 and PM without diesel were highly correlated ($r = 0.99$); these two indicators were not included together in the same regression models. The next highest correlation was observed between traffic and DPM ($r = 0.62$, $p < .0001$). Other relatively high correlations were observed between traffic and TSDFs ($r = 0.55$, $p < .0001$), DPM and TSDFs ($r = 0.52$, $p < .0001$), and DPM and PM2.5 ($r = 0.40$, $p < .0001$).

Results of mixed linear multiple regression models for COVID-19 prevalence are presented in Table 2. Model Set 1 results are for each of the indicators in eight separate models. Significantly higher COVID-19 prevalence was observed in association with PM2.5, DPM and RMP sites when each indicator was considered separately. After removing DPM, the remaining PM2.5 estimate was not associated with the outcome.

Model 2 show results for the emission concentration variables considered simultaneously. In this model, only diesel particulate matter was significantly associated with COVID-19 prevalence. Model 3 shows results for the four pollution sources considered simultaneously. Only RMP sites were significantly associated with COVID-19 prevalence.

Model 4 shows results for all pollution indicators simultaneously. Only diesel particulate matter and RMP sites were significantly associated with higher prevalence risk. The inverse association in Model 4 for traffic is likely an artifact of including DPM and population density in the model, as traffic correlated at $r = 0.62$ with DPM and $r = 0.64$ with population density.

Results of mixed linear multiple regression models for COVID-19 fatality rates are presented in Table 3. Model Set 1 results are for each of the indicators in eight separate models. Individually, significantly higher COVID-19 fatality rates were observed in association with higher PM2.5, diesel particulate matter, and marginally with proximity to TSDFs. The inverse association for traffic is again likely an artifact of including population density in the model. Table 3, Model 2 results are for the emission concentration variables considered simultaneously, and Model 3 results are for the four pollution sources considered simultaneously. The results for Model 2 were the same as for prevalence: higher fatality rates were observed in association with higher diesel particulate matter. For the four sources considered simultaneously (Model 3), only the TSDF variable was significantly associated with higher COVID-19 fatality. Finally, Model 4 showed that diesel particulate matter and TSDFs were significantly associated with higher fatality rates.

As an example of the association of outcomes with covariates, the complete model for DPM (from Table 2 Model 1) with coefficients for covariates is provided in Supplemental Table 2. Higher prevalence was associated with higher percentages of Black, Hispanic and Native American populations. Higher prevalence was also associated with less college education, greater health uninsurance, greater population density, and higher state testing rates. Inverse associations were observed for percent of the population aged 65 and over, and smoking and obesity rates; the inverse age-related association reflects higher prevalence in areas that had generally younger populations, and effects for smoking and obesity may be due to model artifacts after accounting for other covariates.

4. Discussion

Associations observed in a previous COVID-19 study of fatality rates (Wu et al., 2020) for PM2.5 were replicated here for both prevalence and fatality when PM2.5 was considered as an exposure

Table 1
Descriptive statistics by COVID-19 prevalence category.

Variable	COVID-19 Prevalence = 0 (N = 203)				COVID-19 Prevalence >0 to 147.13 (N = 1470)				COVID-19 Prevalence ≥147.13 (N = 1470)			
	Mean	Std Dev	Min.	Max.	Mean	Std Dev	Min.	Max.	Mean	Std Dev	Min.	Max.
COVID Prevalence	0	0	0	0	69.6	38.7	3.76	147	600	826	147	12,641
COVID Prevalence	0	0	0	0	2.04	4.14	0	46.9	25.2	48.8	0	1324
% 65 and over	22.7	6.05	5.97	42.1	20.2	4.58	6.82	41.5	17.9	4.14	4.8	57.6
% Black	1.75	5.70	0	63.0	4.07	6.97	0	61.4	14.9	17.8	0.08	85.4
% Hispanic	9.77	14.9	0.71	81.0	9.07	14.3	0.61	96.4	10.2	13.1	0.64	95.5
% Asian	1.43	4.72	0	43.4	1.23	2.56	0.05	43.0	1.93	2.96	0	35.95
% Native American/PI	5.49	13.6	0	92.2	2.73	7.81	0.13	84.0	1.88	6.59	0.11	92.6
Income Inequality Ratio	4.26	0.88	2.62	8.79	4.46	0.66	2.54	8.69	4.60	0.82	2.99	12.0
% Some College	60.7	13.5	20.4	100	57.4	11.0	15.2	87.8	58.0	12.3	20.9	90.7
% Smokers	15.8	4.16	8.34	41.0	17.6	3.59	7.37	38.7	17.5	3.49	5.91	41.5
% Adults with Obesity	30.3	5.03	17.8	50.5	32.7	4.99	14.4	51.6	33.4	5.83	12.4	57.7
% Uninsured	15.7	5.90	4.92	32.0	13.3	6.24	3.37	42.4	13.7	6.20	2.68	38.7
Population Density (population/square miles)	347	2724	0.05	26,649	81.9	188.6	0.04	2832	355	1544	0.31	484,488
Testing Rate per 100,000	4909	1978	2613	10,609	4646	1554	2613	10,609	4986	1818	26.13	14,584
PM2.5	6.53	1.62	3.36	10.5	8.45	1.83	2.70	12.1	9.19	1.77	3.13	15.1
PM2.5 minus DPM	6.33	1.50	3.34	10.2	8.11	1.73	2.21	11.7	8.67	1.71	2.90	14.5
Ozone	42.9	4.66	28.5	54.7	41.5	4.41	27.2	61.3	41.5	4.36	27.8	64.9
DPM	0.19	0.42	.001	3.50	0.34	0.21	.001	1.78	0.51	0.38	.015	7.0
Traffic	93.1	488	0	4496	85.2	124.7	0	1876	177	328	.008	4444
NPL sites	0.02	0.06	.001	0.74	0.04	0.08	.001	1.26	0.07	0.11	.002	1.08
TSDFs	1.46	12.0	.001	141	0.26	0.40	.002	6.20	0.62	4.53	.004	168
RMP sites	0.38	0.57	.002	3.01	0.46	0.50	.001	2.84	0.56	0.56	.006	4.22

Table 2
Linear multiple regression results for COVID-19 prevalence in association with environmental air pollutants and pollutant sources, adjusting for covariates.[1]

Variable [2]	Model 1 Set [3]	P<	Model 2 [3]	P<	Model 3 [3]	P<	Model 4 [3]	P<
	Estimate (SE)		Estimate (SE)		Estimate (SE)		Estimate (SE)	
PM2.5	23.5 (10.3)	.02	na				na	
Ozone	5.12 (3.29)	.12	3.11 (3.29)	.34			2.36 (3.29)	.47
Diesel PM	253 (50.7)	.001	225 (52.9)	.001			237 (55.8)	.001
PM2.5 minus DPM	15.6 (10.8)	.15	9.80 (10.8)	.36			8.96 (10.8)	.40
Traffic	−0.09 (.06)	.12			−0.10 (.06)	.08	−0.20 (.06)	.02
NPL sites	67.9 (112)	.54			65.6 (112)	.56	−5.59 (113)	.96
TSDFs	−3.63 (4.95)	.46			−3.17 (4.94)	.52	−1.75 (4.95)	.72
RMP sites	75.1 (21.8)	.001			75.9 (21.9)	.001	56.7 (22.6)	.01

1 Covariates included: % population over age 65; percent African American; percent Hispanic; percent Asian; percent Native American/Pacific Islander; income inequality ratio; percent with some college education; percent adult smokers; percent adults with obesity; percent without health insurance; population density; and state testing rate.
2 Abbreviations: PM = particulate matter; DPM = diesel particulate matter; NPL=National Priority List; TSDFS = Treatment, Storage or Disposal Facilities; RMP = Risk Management Plan.
3 In the Model 1 Set, each of the environmental indictors were run in separate models. In Model 2, the pollution emission concentrations were included simultaneously in one model. In Model 3, the pollution sources were included simultaneously in one model. In Model 4, all indictors were considered simultaneously. Note that PM2.5 and PM2.5 minus DPM are not included in the same models.

Table 3
Linear multiple regression results for COVID-19 death rates in association with environmental air pollutants and pollutant sources, adjusting for covariates.[1]

Variable [2]	Model 1 Set [3]	P<	Model 2 [3]	P<	Model 3 [3]	P<	Model 4 [3]	P<
	Estimate (SE)		Estimate (SE)		Estimate (SE)		Estimate (SE)	
PM2.5	1.08 (.54)	.05	na				na	
Ozone	0.26 (.17)	.13	0.12 (.17)	.49			0.10 (.17)	.54
Diesel PM	14.3 (2.54)	.001	15.4 (2.66)	.001			18.7 (2.80)	.001
PM2.5 minus DPM	0.44 (.57)	.44	0.12 (.56)	.84			0.20 (.56)	.72
Traffic	−0.01 (.003)	.002			-.01 (.003)	.001	−0.01 (.003)	.001
NPL sites	5.65 (5.60)	.31			6.92 (5.62)	.21	3.76 (5.65)	.51
TSDFs	0.45 (.25)	.07			0.49 (.24)	.05	0.52 (.25)	.04
RMP sites	0.84 (1.10)	.44			0.97 (1.10)	.38	−0.83 (1.14)	.47

1 Covariates included: % population over age 65; percent African American; percent Hispanic; percent Asian; percent Native American/Pacific Islander; income inequality ratio; percent with some college education; percent adult smokers; percent adults with obesity; percent without health insurance; population density; and state testing rate.
2 Abbreviations: PM = particulate matter; DPM = diesel particulate matter; NPL=National Priority List; TSDFS = Treatment, Storage or Disposal Facilities; RMP = Risk Management Plan.
3 In the Model Set 1, each of the environmental indictors were run in separate models. In Model 2, the pollution emission concentrations were included simultaneously in one model. In Model 3, the pollution sources were included simultaneously in one model. In Model 4, all indictors were considered simultaneously. Note that PM2.5 and PM2.5 minus DPM are not included in the same models.

by itself. However, when considering the effects of diesel PM, the air pollution association with COVID-19 prevalence and fatality was determined specifically by diesel PM and not general PM2.5 when both were based on measurements from prior years potentially representing long-term exposure. Regarding sources, there were different associations dependent on prevalence or fatality as the outcome, with RMP sites showing associations to prevalence, and some evidence for TSDF sites in association with fatality.

A TSDF is a site that receives hazardous solid wastes for treatment, storage or disposal. The hazardous material may be disposed of via landfills, incineration, injection wells, or surface impoundments, kept in waste piles for temporary storage or treatment, or treated as sludge, wastewater or by other means to alter its chemical character (EPA, 2019b, 2019c). A "solid waste" refers to any garbage or refuse; sludge from a wastewater treatment plant, water supply treatment plant or air pollution control facility; and other discarded material resulting from industrial, commercial, mining, and agricultural operations, and from community activities (EPA, 2019b). The EPA states that solid waste is not limited to wastes that are physically solid; solid wastes can be liquid, semi-solid, or contain gaseous material. There are numerous metals, organics and inorganics that are handled by TSDFs, and the current study could not assess associations between different waste materials, treatments or disposal methods and disease outcomes.

Facilities on the NPL, which consist of the most serious uncontrolled or abandoned hazardous waste sites, were not found to be associated with COVID-19. Rather, the associations were observed for the more widespread TSDFs, which are regulated by the EPA and are granted Resource Conservation and Recovery Act permits for the purpose of generating a profit from waste management (EPA, 2005). Additional research on possible health consequences of exposure to TSDFs is warranted.

Risk Management Plans (RMPs) are required for sites that use "extremely hazardous substances" (EPA, 2019d). The EPA lists more than 250 regulated substances that require RMPs depending on the amount used. It appears, however, that little research has been reported on possible public health effects from exposure to these sites. It is also unclear why there would be associations specific to prevalence for one type of site and mortality for another; these findings should be interpreted cautiously and require verification in further research.

Among the exposure measures, associations between diesel PM and COVID-19 prevalence and mortality were observed most strongly and consistently. This particular exposure measure was based on 2014 data, indicating possible effects of long-term exposure but not acute exposure. There is evidence from prior laboratory and epidemiological research that diesel PM impairs lung function and increases susceptibility to viral infection (Castranova et al., 2001; Chauhan and Johnston, 2003; Ciencewicki et al., 2007; Ito et al., 2006). This susceptibility may occur through diesel-induced oxidative stress that increases the density of viral invasion sites in the lungs or by suppressing macrophage function (Ito et al., 2006; Yang et al., 2020).

Limitations of the study include the ecological design. We know population characteristics, exposures and outcomes at the county level but not pollution exposures experienced by individuals with

or without the disease. Measures of pollution emissions and source exposures were based on the most recent EPA public data but they represent conditions in the years 2014–2019. This time differential suggests that long-term exposure to air pollution, especially DPM, could increase susceptibility to COVID-19, but the data do not address acute pollution exposures. Covariates represent a limited set of confounders and may not capture all influences on disease outcomes (Contini and Costabile, 2020). In particular there could be residual confounding such that proximity to exposure sources reflects imperfect adjustment for other demographic or socioeconomic conditions. There may be spatial autocorrelation issues that the current study did not attempt to address, where people in one county may be affected by environmental conditions in adjacent counties. We also did not attempt to address possible effects of the complex lockdown rules that were in effect in some states relative to others and in some cities or counties within states. We examined only a limited set of pollution sources as provided on the EJSCREEN data; other sources, such as power plants or mines, were not examined. We used data on cumulative confirmed cases as of May 31, 2020 and results may change as the disease progresses over time or to different geographic areas. Cases of the disease that were not counted because they were asymptomatic or because of testing shortfalls were not considered; we adjusted for testing rate at the state level but not county level.

In conclusion, results of the study are consistent with prior literature indicating that air pollution increases susceptibility to respiratory infectious illness (Castranova et al., 2001; Chauhan and Johnston, 2003; Jaspers et al., 2005). The evidence suggests that long-term diesel particulate matter exposure may be particularly important. Results also suggest that novel air pollution exposure sources through hazardous waste site TSDFs or RMP sites may be risks for COVID-19 acquisition or fatality. These findings should be taken as exploratory given the ecological design, the time-lag between measures of exposure and outcome, and the uncertainties in complete case counting, and require additional confirmatory research.

CRediT authorship contribution statement

Michael Hendryx: Conceptualization, Methodology, Software, Formal analysis, Writing - original draft, Visualization. **Juhua Luo:** Methodology, Software, Writing - review & editing, Visualization.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.envpol.2020.115126.

Supplemental Table 1
Pearson correlation matrix among the environmental indicators. Correlations at r = .04 or greater are significant at p < .05.

	PM2.5	O3	DPM	PM2.5 minus DPM	Traffic	NPLs	TSDFs	RMPs
PM2.5	1.00							
O3	-.04	1.00						
DPM	.40	.09	1.00					
PM2.5 minus DPM	.99	-.06	.24	1.00				
Traffic	.09	.02	.62	-.02	1.00			
NPLs	.10	.05	.32	.05	.27	1.00		
TSDFs	.05	.01	.52	-.04	.55	.14	1.00	
RMPs	.03	.10	.25	-.02	.18	.11	.05	1.00

Supplemental Table 2
Linear multiple regression results for COVID-19 prevalence in association with environmental air pollutants and pollutant sources, adjusting for covariates.*

Variable	Estimate (SE)	P <
Diesel PM	252.5 (50.6)	.001
% 65 and over	−14.6 (2.83)	.001
% Black	10.5 (1.10)	.001
% Hispanic	3.76 (1.26)	.001
% Asian	−1.10 (4.43)	.80
% Native American/PI	4.54 (1.97)	.02
Income inequality ratio	12.6 (16.9)	.46
% some college	−12.3 (1.38)	.001
% Smokers	−29.8 (6.57)	.001
% Adults with obesity	−7.01 (2.33)	.003
% without health insurance	13.0 (4.02)	.001
Population density	0.09 (.01)	.001
State testing rate	0.05 (.02)	.001

References

Brandt, E.B., Bolcas, P.E., Ruff, B.P., Khurana Hershey, G.K., 2020. Il33 contributes to diesel pollution-mediated increase in experimental asthma severity. Allergy. Epub. https://doi.org/10.1111/all.14181.

Castranova, V., Ma, J.Y., Yang, H.M., Antonini, J.M., Butterworth, L., Barger, M.W., et al., 2001. Effect of exposure to diesel exhaust particles on the susceptibility of the lung to infection. Environ. Health Perspect. 109 (Suppl. 4), 609–612.

Cdc, 2020. Cases of Coronavirus Disease (COVID-19) in the U.S. accessed 06-05-20. https://www.cdc.gov/coronavirus/2019-ncov/cases-updates/cases-in-us.html.

Chauhan, A.J., Johnston, S.L., 2003. Air pollution and infection in respiratory illness. Br. Med. Bull. 68, 95–112.

CHR, 2020. County Health Rankings and Roadmaps. University of Wisconsin and Robert Wood Johnson Foundation, Madison WI accessed 04-07-20. https://www.countyhealthrankings.org/.

Ciencewicki, J., Gowdy, K., Krantz, Q.T., Linak, W.P., Brighton, L., Gilmour, M.I., et al., 2007. Diesel exhaust enhanced susceptibility to influenza infection is associated with decreased surfactant protein expression. Inhal. Toxicol. 19, 1121–1133.

Conticini, E., Frediani, B., Caro, D., 2020. Can atmospheric pollution be considered a co-factor in extremely high level of SARS-CoV-2 lethality in Northern Italy? Environ. Pollut. 114465. https://doi.org/10.1016/j.envpol.2020.114465. Epub.

Contini, D., Costabile, F., 2020. Does air pollution influence covid-19 outbreaks. Atmosphere 11, 377.

COVID Tracking Project, 2020. The Atlantic, Washington DC accessed 06-03-20. https://covidtracking.com/data.

EPA, 2005. Introduction to Treatment, Disposal and Storage Facilities. Environmental Protection Agency, Washington DC accessed 04-23-20. https://www.epa.gov/sites/production/files/2015-07/documents/tsdf05.pdf.

EPA, 2018a. Download EJSCREEN Data. Environmental Protection Agency, Washington DC accessed 04-23-20. https://www.epa.gov/ejscreen/download-ejscreen-data.

EPA, 2018b. Technical Support Document EPA's 2014 National Air Toxics

Assessment. Environmental Protection Agency, Washington DC accessed 04-23-20. https://www.epa.gov/sites/production/files/2018-09/documents/2014_nata_technical_support_document.pdf.
EPA, 2019a. EJSCREEN Technical Documentation. Environmental Protection Agency, Washington, DC accessed 04-23-20. https://www.epa.gov/sites/production/files/2017-09/documents/2017_ejscreen_technical_document.pdf.
EPA, 2019b. Criteria for the Definition of Solid Waste and Solid and Hazardous Waste Exclusions. Environmental Protection Agency, Washington DC accessed 04-23-20. https://www.epa.gov/hw/criteria-definition-solid-waste-and-solid-and-hazardous-waste-exclusions.
EPA, 2019c. National Capacity Assessment Report Persuant to CERCLA Section 104(c)(9). Environmental Protection Agency, Washington DC accessed 04-23-20. https://www.epa.gov/sites/production/files/2019- 12/documents/final_2019_capacity_assessment_report_20191217v1.pdf.
EPA, 2019d. Risk Management Plan (Rmp) Rule. Environmental Protection Agency, Washington DC accessed 06-05-20. https://www.epa.gov/rmp.
Fowler, M., Datson, H., Newberry, J., 2010. Quantitative assessment of dust propagation at a hazardous waste landfill: directional monitoring with elemental analysis. J. Environ. Monit. 12, 879–889.
Gotschi, T., Heinrich, J., Sunyer, J., Kunzli, N., 2008. Long-term effects of ambient air pollution on lung function: a review. Epidemiology 19, 690–701.
Ito, T., Okumura, H., Tsukue, N., Kobayashi, T., Honda, K., Sekizawa, K., 2006. Effect of diesel exhaust particles on mrna expression of viral and bacterial receptors in rat lung epithelial l2 cells. Toxicol. Lett. 165, 66–70.
Jaspers, I., Ciencewicki, J.M., Zhang, W., Brighton, L.E., Carson, J.L., Beck, M.A., et al., 2005. Diesel exhaust enhances influenza virus infections in respiratory epithelial cells. Toxicol. Sci. 85, 990–1002.
JHU, 2020. Cssegisand Data/covid-19. Johns Hopkins University accessed 06-01-2020. https://github.com/CSSEGISandData/COVID-19/blob/master/csse_covid_19_data/csse_covid_19_daily_reports/.
Johnston, J., MacDonald Gibson, J., 2015. Indoor air contamination from hazardous waste sites: improving the evidence base for decision-making. Int. J. Environ. Res. Publ. Health 12, 15040–15057.
Ogen, Y., 2020. Assessing nitrogen dioxide (NO2) levels as a contributing factor to coronavirus (COVID-19) fatality. Sci. Total Environ. 726, 138605.
Porta, D., Milani, S., Lazzarino, A.I., Perucci, C.A., Forastiere, F., 2009. Systematic review of epidemiological studies on health effects associated with management of solid waste. Environ. Health 8, 60.
Takizawa, H., Ohtoshi, T., Kawasaki, S., Abe, S., Sugawara, I., Nakahara, K., et al., 2000. Diesel exhaust particles activate human bronchial epithelial cells to express inflammatory mediators in the airways: a review. Respirology 5, 197–203.
WHO, 2020a. Coronavirus. World Health Organization accessed 04-22-20. https://www.who.int/health-topics/coronavirus#tab=tab_1.
WHO, 2020b. Coronavirus Disease (COVID-19) Pandemic. World Health Organization accessed 04-22-20. https://www.who.int/emergencies/diseases/novel-coronavirus-2019.
Wu, X., Nethery, R.C., Sabath, B., Braun, D., Dominici, F., 2020. Exposure to Air Pollution and COVID-19 Mortality in the United States. medRxiv. https://doi.org/10.1101/2020.04.05.20054502 accessed 04-25-20. https://www.medrxiv.org/content/10.1101/2020.04.05.20054502v1.full.pdf.
Yang, H.M., Antonini, J.M., Barger, M.W., Butterworth, L., Roberts, B.R., Ma, J.K., et al., 2001. Diesel exhaust particles suppress macrophage function and slow the pulmonary clearance of listeria monocytogenes in rats. Environ. Health Perspect. 109, 515–521.
Yang, L., Li, C., Tang, X., 2020. The impact of PM2.5 on the host defense of respiratory system. Front Cell Dev Biol 8, 91.
Zhu, Y., Xie, J., Huang, F., Cao, L., 2020. Association between short-term exposure to air pollution and COVID-19 infection: evidence from China. Sci. Total Environ. 727, 138704.